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DATE: Thursday, February 05, 2004

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<i>DB=PGPB,USPT,JPAB,DWPI; PLUR=YES; OP=ADJ</i>			
<input type="checkbox"/>	L2	(L1 or pacap) and @pd > 20040113	15
<input type="checkbox"/>	L1	(pituitary adenylate cyclase activating polypeptide) and @pd > 20040113	6

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 NEWS 5 SEP 29 DISSABS now available on STN
 NEWS 6 OCT 10 PCTFULL: Two new display fields added
 NEWS 7 OCT 21 BIOSIS file reloaded and enhanced
 NEWS 8 OCT 28 BIOSIS file segment of TOXCENTER reloaded and enhanced
 NEWS 9 NOV 24 MSDS-CCOHS file reloaded
 NEWS 10 DEC 08 CABA reloaded with left truncation
 NEWS 11 DEC 08 IMS file names changed
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 NEWS 13 DEC 09 STN Entry Date available for display in REGISTRY and CA/Caplus
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 NEWS 19 DEC 22 ABI-INFORM now available on STN
 NEWS 20 JAN 27 Source of Registration (SR) information in REGISTRY updated and searchable
 NEWS 21 JAN 27 A new search aid, the Company Name Thesaurus, available in CA/Caplus
 NEWS 22 FEB 05 German (DE) application and patent publication number format changes

NEWS EXPRESS DECEMBER 28 CURRENT WINDOWS VERSION IS V7.00, CURRENT
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=> s pituitary adenylate cyclase activating polypeptide or PACAP
 L1 6494 PITUITARY ADENYLATE CYCLASE ACTIVATING POLYPEPTIDE OR PACAP

=> s l1 and (knockout or transgen? or disrupt? or delet? or deficien?)
 L2 368 L1 AND (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET? OR DEFICIEN?)

)
=> d bib abs

L2 ANSWER 1 OF 366 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
 AN 2004:13351 BIOSIS
 DN PREV200400012847
 TI ***PACAP*** treatment maintains the beta cell mass and retards the onset of hyperglycemia in beta cell-specific calmodulin-overexpressing ***transgenic*** mice.
 AU Tsunekawa, S. [Reprint Author]; Miura, Y. [Reprint Author]; Yamamoto, N. [Reprint Author]; Ariyoshi, Y. [Reprint Author]; Senda, T.; Oiso, Y. [Reprint Author]; Niki, I.
 CS First Dept of Internal Medicine, Nagoya University, School of Medicine, Nagoya, Japan
 SO Diabetes & Metabolism, (August 2003) Vol. 29, No. Hors serie 2, pp. 4S58.
 print.
 Meeting Info.: 18th International Diabetes Federation Congress. Paris, France. August 24-29, 2003.
 ISSN: 1262-3638.
 DT Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LA English
 ED Entered STN: 24 Dec 2003
 Last Updated on STN: 24 Dec 2003

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FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 16:51:20 ON 05 FEB 2004
 L1 6494 S PITUITARY ADENYLATE CYCLASE ACTIVATING POLYPEPTIDE OR PACAP
 L2 368 S L1 AND (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET? OR DEFICIEN?)

=> s l1 (1s) (knockout or transgen? or disrupt? or delet? or deficien?)
 L3 280 L1 (1S) (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET? OR DEFICIEN?)

=> dup rem l3
 PROCESSING COMPLETED FOR L3
 L4 154 DUP REM L3 (126 DUPLICATES REMOVED)

=> s l4 and PY=2002
 L5 35 L4 AND PY=2002

=> s l4 and PY<=2002
 2 FILES SEARCHED...
 L6 124 L4 AND PY<=2002

=> d his

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 L2 368 S L1 AND (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET? OR DEFICIEN?)
 L3 280 S L1 (1S) (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET? OR DEFICIEN?)
 L4 154 DUP REM L3 (126 DUPLICATES REMOVED)
 L5 35 S L4 AND PY=2002
 L6 124 S L4 AND PY<=2002

=> s l2 and psychiatrist?
 L7 3 L2 AND PSYCHIATR?

=> dup rem l7
 PROCESSING COMPLETED FOR L7
 L8 3 DUP REM L7 (0 DUPLICATES REMOVED)

=> d bib abs 1-
 YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):
 YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L8 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2004 ACS on STN
 AN 2003:1004671 CAPLUS
 DN 140:3795
 TI Non-human animal model for ***psychiatric*** disorder with ***deficient*** in function of ***pituitary*** ***adenylate*** ***cyclase*** - ***activating*** ***polypeptide*** gene
 IN Baba, Akemichi; Matsuda, Toshio; Hashimoto, Hitoshi; Shintani, Norihito
 PA Japan
 SO U.S. Pat. Appl. Publ., 13 pp., Cont.-in-part of U.S. Pat. Appl. 2001
 34,885.
 CODEN: USXXCO
 DT Patent
 LA English

FAN.CNT 2
PATENT NO. KIND DATE APPLICATION NO. DATE

PI US 2002162128 A1 20021031 US 2002-73135 20020213
US 2001034885 A1 20011025 US 2001-835627 20010417
PRAI JP 2000-118088 A 20000419
US 2001-835627 B2 20010417

AB The invention relates to mammalian model animal for ***psychiatric*** disorders having a chromosome of a somatic cell and a germ cell with ***deficiency*** of function of ***pituitary*** ***adenylate*** ***cyclase*** - ***activating*** ***polypeptide*** (***PACAP***) gene. The exon 5 of gene ***PACAP*** of mammal is ***disrupted*** and replaced with neomycin resistance gene. The results of behavioral expts. with ***PACAP*** -/- mice demonstrate that ***disruption*** of the ***PACAP*** gene in mice lead to perturbations in psychomotor behaviors, esp. the exploratory component of locomotor behavior, implicating ***PACAP*** in psychotic brain functions. Furthermore, the 5-HIAA level was decreased slightly in the cortex and striatum of the ***PACAP*** -/- mouse brain. One of the striking findings of the present study was that ***PACAP*** -/- mice showed abnormal jumping behavior in the open field arena. The ***PACAP*** -/- mouse should be a valuable tool to investigate both normal and pathol. processes in which ***PACAP*** has been proposed to play a role.

L8 ANSWER 2 OF 3 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:587826 BIOSIS
DN PREV200200587826

TI Higher brain functions of ***PACAP*** and a homologous Drosophila memory gene amnesiac: Insights from knockouts and mutants.
AU Hashimoto, Hitoshi; Shintani, Norihito; Baba, Akemichi [Reprint author]
CS Laboratory of Molecular Neuropharmacology, Graduate School of Pharmaceutical Sciences, Osaka University, 1-6 Yamadaoka, Suita, Osaka, 565-0871, Japan
bab@phs.osaka-u.ac.jp
SO Biochemical and Biophysical Research Communications, (September 27, 2002)
Vol. 297, No. 3, pp. 427-432. print.
CODEN: BBRCA9. ISSN: 0006-291X.

DT Article
LA English
ED Entered STN: 13 Nov 2002
Last Updated on STN: 13 Nov 2002

AB Neuropeptides usually exert a long-lived modulatory effect on the small-molecule neurotransmitters with which they colocalize via regulation of the response times of second messenger systems. ***Pituitary*** ***adenylate*** ***cyclase*** - ***activating*** ***polypeptide*** (***PACAP***) functions as a neuromodulator and neurotransmitter and regulates a variety of physiological processes. ***PACAP*** is structurally highly conserved during evolution, implying its vital importance. In Drosophila, loss-of-function mutations in a ***PACAP*** -like neuropeptide gene, amnesiac (amn), affect both memory retention and ethanol sensitivity. The amnesiac gene is expressed in neurons innervating the mushroom body lobes, the olfactory associative learning center. Conditional genetic ablation of neurotransmitter release from these neurons mimics the amnesia memory phenotypes, suggesting an acute role for amnesiac in memory. However, genetic rescue experiments also suggest developmental defects in amnesiac mutants, implying a role in neuronal development. There is a parallel between memory formation in Drosophila and mammals. ***PACAP*** -specific (PAC1) receptor- ***deficient*** mice show a deficit in hippocampus-dependent associative learning and mossy fiber long-term potentiation (LTP). Meanwhile, ***PACAP*** - ***deficient*** mice display a high early mortality rate and additional CNS phenotypes including behavioral and psychological phenotypes (e.g., hyperlocomotion, intense novelty-seeking behavior, and explosive jumping). A functional comparison between ***PACAP*** and amnesiac underlines phylogenetically conserved functions across phyla and may provide insights into the possible mechanisms of action and evolution of this neuropeptidergic system.

L8 ANSWER 3 OF 3 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1999:28142 BIOSIS
DN PREV199900028142

TI Truncated presenilin 2 derived from differentially spliced mRNAs does not affect the ratio of amyloid beta-peptide 1-42/1-40.
AU Gruenberg, Juergen; Walter, Jochen; Eckman, Chris; Capell, Anja; Schindzielorz, Alice; Younkin, Steven; Mehta, Nitin; Hardy, John; Haass, Christian [Reprint author]
CS Central Inst. Mental Health, Dep. Molecular Biol., J5, 68159 Mannheim, Germany
SO Neuroreport, (Oct. 5, 1998) Vol. 9, No. 14, pp. 3293-3299. print.
CODEN: NERPEZ. ISSN: 0959-4965.

DT Article
LA English
ED Entered STN: 3 Feb 1999
Last Updated on STN: 3 Feb 1999

AB Numerous mutations in the presenilin (PS) genes cause early onset familial Alzheimer's disease (FAD). Here we characterize the expression of two naturally occurring alternative PS2 transcripts which lack either exons 3 and 4 (PS2 DELTAexon3,4) or exons 3, 4, and 8 (PS2 DELTAexon3,4,8). These transcripts do not contain the natural initiation codon within exon 3.

The transcripts are efficiently translated as N-terminal truncated proteins. These ***deleted*** proteins are still able to regulate formation of endogenous PS fragments, indicating that the C-terminal half of the PS2-protein is sufficient for this phenomenon. Although approx 50% of the PS1 and both PS2 mutations occur within the N-terminal region lacking in the PS2 DELTAexon3,4 and PS2 DELTAexon3,4,8 proteins, expression of these truncated proteins does not affect pathological generation of amyloid beta-peptide (Abeta). This suggests that point mutations causing AD are gain of function mutations.

=> d his

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L1 8494 S PITUITARY ADENYLATE CYCLASE ACTIVATING POLYPEPTIDE OR PACAP
L2 386 S L1 AND (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET? OR DEFICIENCY)
L3 280 S L1 (15) (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET? OR DEFICIENCY)
L4 154 DUP REM L3 (126 DUPLICATES REMOVED)
L5 35 S L4 AND PY<=2002
L6 124 S L4 AND PY<=2002
L7 3 S L2 AND PSYCHIATR?
L8 3 DUP REM L7 (0 DUPLICATES REMOVED)

=> s L2 and homozygo?

L9 7 L2 AND HOMOZYGO?

=> dup rem l9

PROCESSING COMPLETED FOR L9

L10 4 DUP REM L9 (3 DUPLICATES REMOVED)

=> d bib abs 1-

YOU HAVE REQUESTED DATA FROM 4 ANSWERS - CONTINUE? Y/(N):y

L10 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:282277 CAPLUS
DN 138:282471

TI Use of human and mouse insulin 6 gene-encoded protein in improving spermatoocyte motility in diagnosis and treatment of male sterility
IN Menon, Ram K.; Sperling, Mark A.; Lu, Chunxia; Witchel, Selma; Kasik, John PA Children's Hospital of Pittsburgh, USA
SO PCT Int. Appl., 92 pp.
CODEN: PIXXD2

DT Patent
LA English
FAN.CNT 1
PATENT NO. KIND DATE APPLICATION NO. DATE

PI WO 2003028457 A1 20030410 WO 2002-US30781 20020927
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2003229035 A1 20031211 US 2001-987399 20010928

PRAI US 2001-987399 A 20010928
AB The present invention relates to a novel gene from the insulin family, INS6, which expresses a protein restoring motility in ciliated cells. The proteins of the insulin family play essential roles in pleiotropic physiol. processes affecting metab., growth, and reprodn. A new member of the insulin family named Ins16 is disclosed playing an essential role in ciliated cell activity. Ins16 plays an essential role in spermatoocyte function. Thus, the INS6 gene and its protein product are useful in the treatment of infertility caused by the loss of spermatoocyte motility. A method of modulating male fertility is disclosed.

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 2 OF 4 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:504687 BIOSIS
DN PREV200100504687

TI Sympathoadrenalin function in ***pituitary*** ***adenylate*** ***cyclase*** - ***activating*** ***polypeptide*** (***PACAP***)- ***deficient*** mice.

AU Hamelink, C. R. [Reprint author]; Lee, H. W. [Reprint author]; Damadjic, R. [Reprint author]; Tjurmina, O.; Young, W. S. [Reprint author]; Weihe, E.; Eiden, L. E. [Reprint author]

CS Lab. of Cellular and Molecular Regulation, NIMH, NIH, Bethesda, MD, USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 620. print.

Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 31 Oct 2001
Last Updated on STN: 23 Feb 2002
AB ***PACAP*** 's role as a splanchnic neurotransmitter regulating adrenomedullary secretion is imprecisely defined. We generated ES cells heterozygous for ***PACAP*** "deletion" by homologous recombination, and from them, mice ***homozygous*** for the wild-type (+/+ or null (-) ***PACAP*** allele. Challenge with 2-5 U/kg of insulin resulted in decreased survival, a less profound elevation of circulating epinephrine, and a more profound hypoglycemia, in (-) than in (+/) mice. Decreased survival of (-) mice after insulin challenge could be partially reversed by concomitant administration of glucose (20ug/mouse/hour, i.p.), isoproterenol (3ug/mouse/hour, i.p.), or ***PACAP*** (10nmol/mouse single dose, i.p.) with 5 U/kg insulin (i.p.). In addition to decreased epinephrine output in ***PACAP*** (-) mice following insulin, ***PACAP*** (-) mice exhibited no elevation in the activity of adrenal tyrosine hydroxylase, the rate-limiting enzyme in catecholamine biosynthesis, whereas adrenal tyrosine hydroxylase activity was doubled 4-8 hours after insulin administration (2 U/kg) in ***PACAP*** (+/+) mice. These data suggest that ***PACAP*** is required to couple secretion and biosynthesis of adrenomedullary catecholamines to maintain plasma catecholamine levels sufficient for gluconeogenesis during prolonged hypoglycemia.

L10 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

DUPLICATE 1
AN 2000:491720 BIOSIS
DN PREV200000491841
TI ***Pituitary*** ***adenylate*** ***cyclase*** -
activating ***polypeptide*** precursor is processed solely by
prohormone convertase 4 in the gonads.
AU Li, Min [Reprint author]; Mibkay, Majambu; Arimura, Akira
CS U.S.-Japan Biomedical Research Laboratories, Tulane University Hebert
Center, 3705 Main Street, Belle Chasse, LA, 70037-3001, USA
SO Endocrinology, (October, 2000) Vol. 141, No. 10, pp. 3723-3730. print.
CODEN: ENDOAO ISSN: 0013-7227.

DT Article
LA English
ED Entered STN: 15 Nov 2000
Last Updated on STN: 10 Jan 2002

AB ***Pituitary*** ***adenylate*** ***cyclase*** -
activating ***polypeptide*** (***PACAP***) is abundant not
only in the brain, but also in the testis. Immunohistochemical studies
have shown that ***PACAP*** -LI in rat testis is expressed stage
specifically in spermatids. This suggests that testicular ***PACAP***
participates in the regulatory mechanism of spermatogenesis.
Additionally, the ovary contains a relatively small amount of
PACAP , conceivably involved in the regulation of folliculogenesis.
PACAP is synthesized as a prohormone and is processed by
prohormone convertases, such as PC1, PC2, and PC4. PC4 is expressed only
in the testis and ovary, where neither PC1 nor PC2 is expressed. However,
whether PC4 is the sole endoprotease for the ***PACAP*** precursor in
the gonads remains unknown. Recent studies using PC4- ***transgenic***
mice revealed that male PC4-null mice exhibited severely impaired
fertility, although spermatogenesis appeared to be normal. The female
PC4-null mice exhibited delayed folliculogenesis in the ovaries. To
examine whether PC4 is the sole processing enzyme for the ***PACAP***
precursor in the gonads, we analyzed testicular and ovarian extracts from
the PC4-null and wild-type mice for ***PACAP*** (PACAP38 and PACAP27)
and its messenger RNA using reverse phase HPLC combined with specific RIAs
and ribonuclease protection assay, respectively. For RIAs, three
different polyclonal antisera with different recognition sites were used
to identify PACAP38, PACAP27, and its precursor. Neither the testis nor
the ovary from the PC4-null mice expressed PACAP38 or PACAP27, but the
levels of ***PACAP*** transcripts in the testis and ovary of
homozygous PC4- ***deficient*** mice were considerably elevated
compared with those of the wild-type and heterozygous animals. The
findings indicate that PC4 is the sole processing enzyme for the precursor of
PACAP in the testis and ovary of mice. The possibility that
the absence of bioactive ***PACAP*** in the testis and ovary of
PC4-null mice caused severely impaired fertility in the males and delayed
folliculogenesis in females warrants investigation.

L10 ANSWER 4 OF 4 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

DUPLICATE 2
AN 1995:34748 BIOSIS
DN PREV199598049046
TI Molecular Basis of Familial Growth Hormone ***Deficiency*** .
AU Perez Jurado, L. A.; Argente, J. [Reprint author]
CS Div. Paediatr. Endocrinol., Hosp. Nino Jesus, Avda. Menendez y Pelayo, 65,
E-28009 Madrid, Spain
SO Hormone Research (Basel), (1994) Vol. 42, No. 4-5, pp. 189-197.
CODEN: HRMRA3. ISSN: 0301-0163.

DT Article
General Review, (Literature Review)
LA English
ED Entered STN: 25 Jan 1995
Last Updated on STN: 14 Mar 1995
AB A significant proportion of cases of GH ***deficiency*** (5-30%) may

be due to genetic causes. At least four Mendelian types of isolated GH deficiency (IGHD) have been delineated based on the mode of inheritance and the degree of GH deficiency: IGHDA type IA, autosomal recessive with absent endogenous GH; type IB, autosomal recessive with diminished GH; type II, autosomal dominant with diminished GH; and type III, X-linked with diminished GH. Most patients with IGHDA type IA have heterogeneous deletions, ranging in size from 6.7 kb to 45 kb, that encompass the entire gene encoding for pituitary GH. GH-1. Nonsense, frameshift and splice GH-1 mutations that predict a complete lack of bioactive GH synthesis in homozygotes have also been reported in association with IGHDA IA. Additionally, some cases of IGHDA type II have dominant negative mutations in one allele of the GH-1 gene. Panhypopituitary Dwarfism (PD), a condition characterized by GH deficiency of at least other pituitary trophic hormone in addition to GH deficiency, can have autosomal and X-linked modes of inheritance. Interestingly, both recessive and dominant mutations at the gene encoding for the pituitary transcription factor Pit-1 have been found in a specific subtype of PD that combines GH, prolactin and TSH deficiencies. In contrast, the loci and mutations responsible for the other Mendelian forms of IGHDA and PD remain unknown. Linkage studies using genetic markers have excluded the GH locus on chromosome 17 in approximately 50% of the cases and the GH-releasing hormone (GHRH) locus on chromosome 9.

chromosome 20 in all the studied families (types IB and II) in whom the mutation cannot be traced to defects in these genes. Furthermore, several uncharacterized loci on the X chromosome must be required for normal GH secretion. In summary, genetic studies have provided a better understanding of the mechanism of GH "deficiency", as well as new tools for specific diagnosis of several forms of IGHD and PD. However, isolation and evaluation of other genes involved in GH secretion is still necessary. Several possible candidate genes have been recently cloned and characterized, including genes encoding the human GHRH receptor, the "pituitary" "adenylate" "cyclase" "activating" "polypeptide" ("PACAP") and the "PACAP" receptor. Analysis of these genes in IGHD and PD families may clarify the molecular basis of the defect and also provide new insights into the complex regulation of GH.

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